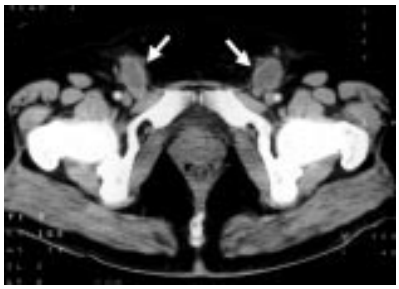

Figure 2 Pelvic CT scan showing bilateral inguinal lymphadenopathy.

Vulval cancer accounts for 3–5% of female genital tract malignancies. Risk factors include lichen sclerosus, vulval intraepithelial neoplasia, and infection with oncogenic human papillomavirus (HPV) types.[1] STDs other than HPV are also associated with an increase in the risk of developing vulval neoplasia.[2] The presence of antibodies to HSV type 2 has been implicated as a risk for cervical pathology[3] but a role for HSV in vulval neoplasia is unclear. Vulval basal cell carcinoma presenting as culture negative genital herpes has been reported.[4] In our case the carcinoma was culture positive for HSV; this may have been due to new infection or to reactivation of pre-existing HSV in the presence of malignancy. This case highlights the need for biopsy of herpetic lesions which fail to respond to standard therapy.

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Accepted for publication 12 January 2001

Sexually shared infections

EDITOR,—Those who have spent some time in genitourinary medicine will surely agree that the specialty has gone through vast changes over the years. Not only the nomenclature of our clinics from VD clinics or special clinics to psychosexual health departments but also the name of our specialty itself has gone through a metamorphosis.

I was therefore interested to note the term "sexually shared infections" suggested by Hopwood *et al*[1] and wondered what message it would project to our patients, sorry our "clients." Hence, I decided to test this new term in my clinic and would like to share the results with the readers of *STI*.

Firstly, I saw a young girl who had primary presentation of genital warts. I suggested that she might have "shared" this infection with her partner to which she replied, "Look doctor, I know HE gave it to me because he is the one who was sleeping around."

The next one was a young man who presented with acute gonorrhoea. When I said he might have shared this infection with the one night stand he had in Manchester he replied, "Look doctor, I am no fool. I was so drunk that night that I couldn't perform but she went ahead anyway then this happened."

The third one was a chlamydia reinfection. The young girl was found to be positive and received a single dose regimen. Her boyfriend was referred to a GUM clinic but by the time he attended they had had protected sex but the condom split and the girl was reinfected. When I mentioned the "shared" element she fumed, "It was him who gave me this in the first place and he wouldn't get treatment himself because he felt OK."

English is not my first language but I always thought that you "share" something that is nice. Like sharing the tender moments, sharing your cake, British Airways share offer when it floated on stock market, etc.

Sharing an STI to me sounds a bit awkward.

In my opinion people transmit the infections knowingly or unknowingly because of their high risk sexual behaviour. It does not matter if we try to play this down and make it acceptable. There always will be some stigma attached to STIs but we should ensure awareness, patient education, and partner notification. I believe this should be done by professionals in a confidential setting in a genitourinary medicine clinic. Changing the terminology about the mode of transmission will not eliminate the stigma attached to STIs but the more open we are about infections the better it will be for our patients.

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Deterioration of disseminated cutaneous *Mycobacterium avium* complex infection with a leukaemoid reaction following institution of highly active antiretroviral therapy

EDITOR,—The impact of highly active antiretroviral therapy (HAART) on the incidence of opportunistic infections (OI) in HIV infected patients has been well documented. HAART also frequently alters the clinical course of OI. Increasingly, immune reconstitution disease is recognised after starting HAART in patients with latent or established OI.[1–3] Despite the marked reduction in incidence of disease due to *Mycobacterium avium* complex (MAC) in the HIV infected population over the past 5 years, this OI is often implicated in immune reconstitution disease and may be difficult to treat.[1 3] Focal mycobacterial lymphadenitis appears to be the commonest manifestation,[1 3] but other organs may be involved.

A 40 year old white HIV positive man presented with *Staphylococcus aureus* tricuspid valve endocarditis: blood cultures also grew MAC. He had a history of cutaneous

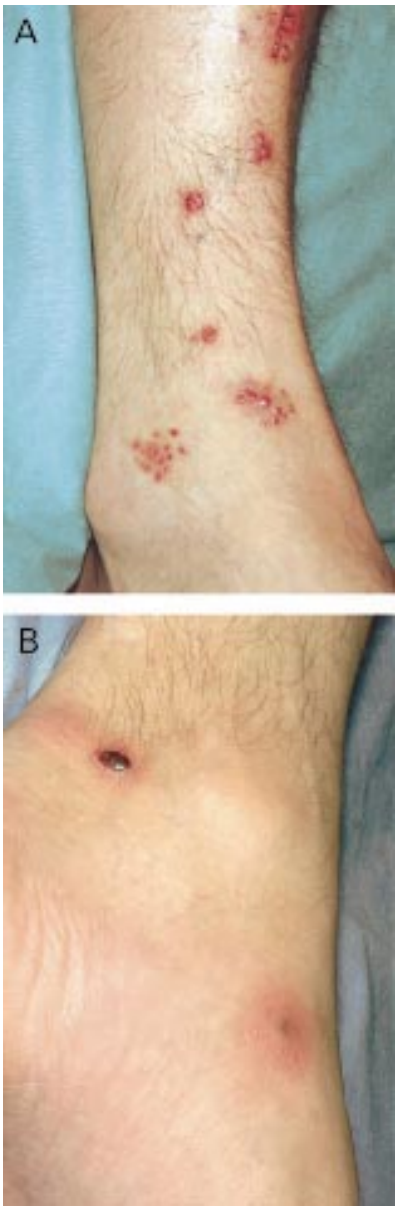

Figure 1 (A) At initial presentation with MAC infection. Patient's right shin and ankle showing painless dermal papules and nodules. A skin biopsy has been performed on the right shin. (B) Five days after re-presentation. Medial aspect of left ankle. There are two erythematous lesions, which were tender to touch. Both have a pustular centre.

Kaposi's sarcoma and oesophageal candidiasis. After inpatient treatment of the endocarditis he defaulted from outpatient follow up. Five months later he re-presented with a 3 month history of fever, cough, malaise, and painless skin lesions on both arms and legs. Examination showed multiple dermal papules and nodules with necrosis and some scarring (fig 1A). The CD4 count was 10 cells $\times 10^6$/l and the HIV viral load 202 300 copies/ml. Skin biopsy revealed multiple poorly formed granulomata; numerous acid fast bacilli (AFB) were seen and MAC was subsequently cultured from skin, sputum, urine, and blood. He was treated with rifabutin, clarithromycin, ethambutol, and isoniazid; treatment was reduced to clarithromycin and ethambutol alone, after 6 weeks when the mycobacterium was speciated. HAART,